RELATIONSHIP BETWEEN INTRAVESICAL PROSTATIC PROTRUSION (IPP) AND THE POSTOPERATIVE OUTCOMES IN PATIENTS WITH BENIGN PROSTATIC HYPERPLASIA

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CERTIFICATE

This is to certify that this dissertation entitled **" RELATIONSHIP BETWEEN INTRAVESICAL PROSTATIC PROTRUSION AND THE POSTOPERATIVE OUTCOMES IN PATIENTS WITH BENIGN PROSTATIC HYPERPLASIA "** submitted by

Dr. R. SRIVATHSAN appearing for **M.Ch (Urology)** degree examination in August 2014 is a original bonafide record of work done by him during the academic period of August 2011 to July 2014 under direct supervision and guidance in partial fulfillment of requirement of the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamilnadu, India.

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Dr. R. SRIVATHSAN appearing for **M.Ch UROLOGY** degree examination in August 2014 is an original bonafide record of work done by him during the academic period of August 2011 to July 2014 under my guidance and supervision in partial fulfillment of requirement of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, Tamil Nadu, India.

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I, **Dr. R. Srivathsan ,** solemnly declare that this dissertation titled **"RELATIONSHIP BETWEEN INTRAVESICAL PROSTATIC PROTRUSION AND THE POSTOPERATIVE OUTCOMES IN PATIENTS WITH BENIGN PROSTATIC HYPERPLASIA"** was done by me in the Department of Urology, Kilpauk Medical College Hospital and Government Royapettah Hospital , Chennai under the guidance and supervision of **Dr.C.Ilamparuthi, M.S.,M.Ch.,DNB.,** Professor of Urology, Government Royapettah Hospital.

This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical University, Chennai-600032 in partial fulfilment of the University requirements for the award of the degree of M.Ch., Urology.

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ABBREVIATIONS

AUA American Urological Association

BPE Benign Prostatic Enlargement

BPH Benign Prostatic Hyperplasia

AUR Acute urinary retention

BOOI Bladder outlet obstruction Index

DRE Digital Rectal Examination

LUTS Lower Urinary Tract symptoms

TRUS Transrectal Ultrasonography

TAUS Transabdominal Ultrasonography

TPV Total prostate volume

TZV Transitional zone volume

TZI Transitional zone index

IPP / IPPV Intravesical prostatic protrusion

DWT Detrusor wall thickness

UEBW Ultrasonic estimation of bladder weight.

PVR	Postvoid residue
PSA	Prostate specific antigen
Qmax	Maximum flow rate
Pdet	Detrusor pressure
QoL	Quality Of Life
DHT	Dihydrotestosterone

INTRODUCTION

The American Urological Association introduced an initiative to identify and define the research priorities in urology. National Urology Research Agenda (NURA) defines the priority issues in urology and identified BPH as a field for research[1].

Benign prostatic hyperplasia (BPH) is a very frequent condition affecting men of old age. It is seen in about 10 percent of men of the age of less than 40, and jumps to 80 percent in age group of 80 years. Inspite of various other causes now being considered, Benign prostatic hyperplasia still remains one of the most common benign diseases in men that can lead to lower urinary tract symptoms, with or without bladder outlet obstruction (BOO).A multicenter study performed showed that the age-related division of men with symptoms was higher in the Asia Pacific than those in Western countries [2]. The reason behind this incidence is yet to be unraveled.

BPH is a hyperplastic process (and not hypertrophy) involving both the stromal and glandular elements of the prostate. BPH significantly affects the quality of life in many of the patients. 50 to 70% of men with histological features of BPH also have a prostate volume of more than 25 ml, and up to 28% have moderate to severe LUTS [3,4]. Even though most seek medical intervention because of bothersome symptoms, BOO was

found in 60% in those symptomatic and 52% in those asymptomatic[6, 7.] Lower urinary tract symptoms decrease the patient's QOL. Bothersome LUTS needing intervention can occur in up to 30% of men who are older than 65 years [5].

Several theories are proposed in the etiopathogenesis of BPH. These include

- Age-related tissue changes,
- Metabolic syndrome
- Hormonal alterations,
- Inflammation[8].

Although androgens do not cause BPH, it has been postulated that the presence of androgens is essential for pathogenesis of the same. Also the association between metabolic syndrome and the development of BPH should be borne in mind. Finally, recent increasing evidence seems to suggest the idea that BPH arises as a result of an inflammatory-based disorder.

TURP is the second most frequent surgical intervention [9] that a male of age greater than 50 years undergoes, second only to cataract surgery. TURP has withstood the test of times in being the gold standard treatment in the management of BPH[2] and even in some cases of carcinoma prostate as a channel creating procedure. The advent of

LASERs in endourology has put the exclusivity of TURP in the management of BPH in jeopardy. Holmium laser (HoLEP) is currently taken to be the gold standard procedure [13,14] though it is still questioned by many urologists. However in developing countries the prohibitive cost of these lasers make their widespread public use difficult.

TURP still remains the widely used technique for the management of BPH[10,11,12].

TURP, with the advent of newer technologies in diathermy and visual scopes, has turned from a complication fraught procedure to a relatively safe one. But still the risks of TURP syndrome and dyselectrolytemia do exist especially in high-risk cardiac patients accentuated by the use of glycine as airrigant fluid. The advent of bipolar diathermy has made the use of normal saline as airrigant in the procedure safe.

IPP occurs as the prostate gland enlarges along the region of least resistance i.e. into the bladder. This is predominantly due to growth of the median lobe and maybe associated with enlargement of the lateral lobes. It was proposed that this IVP leads to a ball valve and thereby leading to obstruction. This in turn disrupts the funneling of the bladder neck increasing urethral resistance. This leads to the dyskinetic movement of the bladder during micturition reflex [15,16,18,19,21]

Nose[22] et al. was the first proponent who studied if there is any correlation between IPP and the Bladder outlet obstruction index and found that grading of IPP based on the 10mm criteria correlated well with the BOOI.

Keqin[20]et al. proposed a new classification of IPP grading (< 5mm, 5 -10 mm, > 10mm) and found that this IPP grading correlated well with various other parameters (TPV, PVR, PSA, Qmax, BOOI, PdetQmax) as well as the probability of AUR, bladder wall thickening, detrusor hypercontractlity, and bladder compliance.

Ku[23] et al. found that the BOOI was significantly greater in patients with IPP > 10mm than in those who had minimal IPP.

Mariappan[17] et al. showed that the decatheterisation trial is likely to fail if IPP > 10 mm. IPP's correlation with prostate volume, obstructive symptoms and PFR suggested that it does have clinical significance in predicting the need for intervention.

AIM AND OBJECTIVE

To compare the outcomes of TURP in cases with and without significant IPP (Intravesical prostatic Protrusion)

REVIEW OF LITERATURE

Sex accessory tissues which includes prostate, seminal vesicles, ampullary glands, and bulbourethral glands, are believed to play a major but not fully understood role in the reproductive process. The presence of the prostate is found to be universal in all mammals[28,29].

The prostate first appears and starts its development from the urogenital sinus during the third month of fetal growth, and development is directed primarily by DHT[30,31]. The prostate is formed by five epithelial buds on the posterior side of the urogenital sinus which form the outer surface, on either side of the verumontanum, which then invades the mesenchyme. The top buds which form the inner zone are mesodermal in origin and the lower buds which form the outer zone are endodermal in origin. This is of potential importance since the inner zone gives rise to benign prostatic hyperplasia (BPH) tissue, whereas the outer zone contains the origin of cancerous tissue. These two zones develop as concentric circles around the urethra. The long branched ducts along the outside of this zone form the thick outer layer of the true prostate gland. The center portion of the prostate contains the mucosal and submucosal gland and the ejaculatory ducts as well as the small remnants of the müllerian duct—the utriculusprostaticus, which forms the small prostatic

utricle. The prostate is well differentiated by the fourth month of fetal development.The prostate forms acini and collecting ducts by arborization into the urethra; the growth occurs primarily on the tips, as the ducts extend and branch during development. This concept that dynamic growth processes occur along a budding and branching system was developed from studies on the mouse and rat prostate [30](Sugimura et al, 1986 ; Banerjee et al, 1993a, 1993b; Cunha, 1994).

A normal prostate weighs 18 grams and has an anterior, posterior and lateral surface. The prostatic urethra traverses through it. Its got a narrow apex directed inferiorly and a broad base superiorly.[35,38]. Its capsule is composed of collagen, elastin, and smooth muscle. Posterolaterally, the capsule has an average thickness of 0.5 mm. The loose areolar tissue defines the thin plane between Denonvilliers' fascia and the rectum. The anterior and anterolateral surface capsule blends with the endopelvic fascia. The puboprostatic ligaments fix the apex to thepubic bone

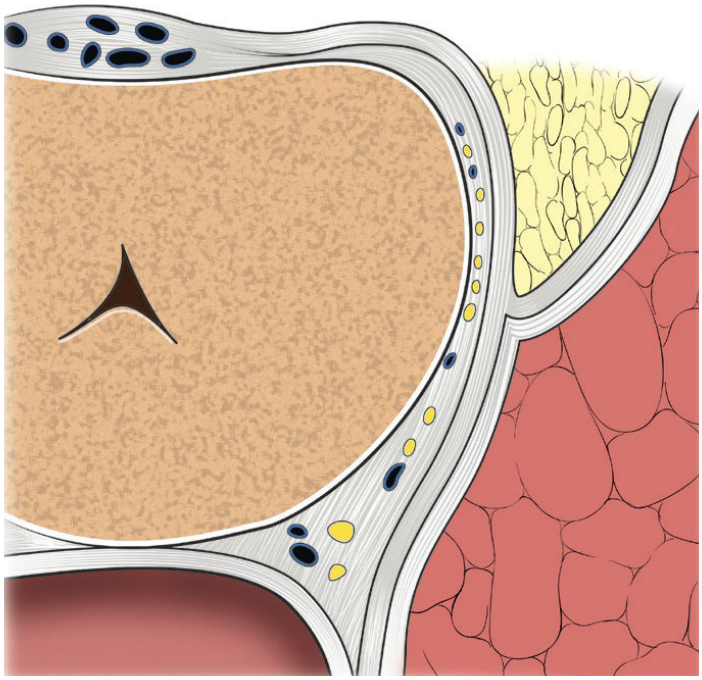

Figure 1 Anatomy of Prostate gland

Laterally, the pubococcygeal portion of levatorani muscle covers it is related to its endopelvic fascia. Below is the Myers lateralendoprostatic fascia.[32,33]. The cavernosal nerves run posterolateraly the cavernosal veins run to the prostate in the lateral prostatic fascia.

The apex of the prostate runs continuous with the striated rhabdosphincter[39,40]. Histologically, normal prostatic glands extend into the rhabdosphincter with no intervening capsule.

Structure

Prostate has 70% glandular elements and 30% fibromuscularstroma. The stroma encircles the glands of the prostate and

its contraction during ejaculation express prostatic secretions into the urethra.

ZONAL ANATOMY[34] :

1) The five lobes of the prostate can be seen prior to twenty weeks gestation.

2) 3 lobes are recognizable, 2 lateral and a median lobes

3) From a pathological viewpoint, the glandular tissue may be subdivided into 3 distinct zones.

1) Peripheral (70% by volume)-PZ

2) Central (25% by volume)-CZ

3) Transition (5% by volume)-TZ

4) Fibromuscularstromafills the space between the peripheral zones anterior to the preprostatic urethra.

5) TheCZ, which surrounds the ejaculatory ducts, is posterior to the preprostatic urethra and is conical in shape.

6) Mucus secreting glands are in the tissue around the preprostatic urethra

7) The central zone is rarely involved in pathological disease as it shares different histological properties from the rest of the prostate and is thought to be derived from the Wolffian duct system (much like the epididymis, vasa deferentia and seminal vesicles)

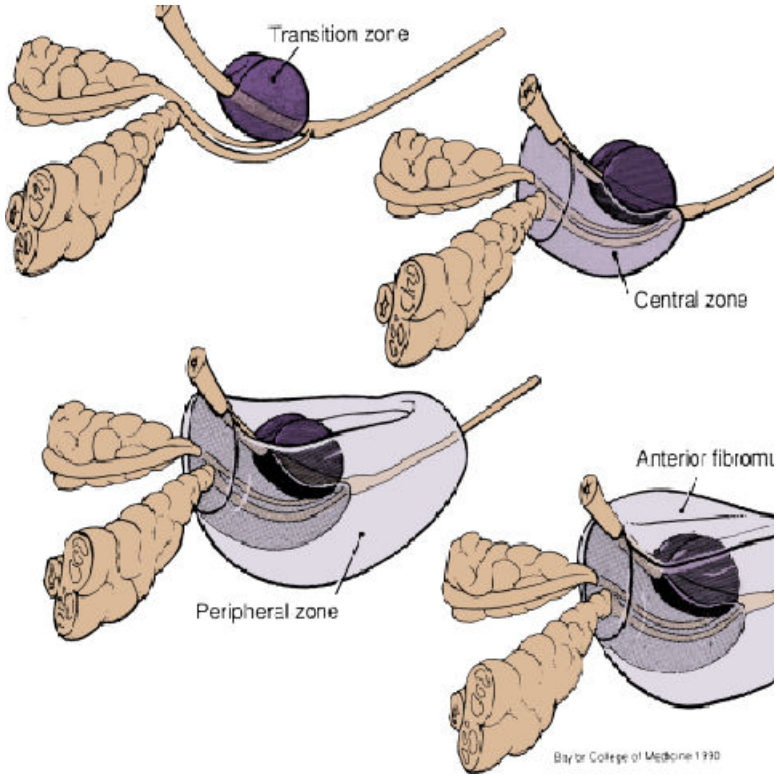

Figure 2

Zonal Anatomy of Prostate

Clinically the prostate has two lateral lobes and a median lobe. The lateral lobes are separated by a central sulcus that is palpable on per rectal examination.

Vascular Supply

The arterial supply is from the inferior vesical artery.

ETIOPATHOGENESIS OF BPH

Multiple theories have been implicated in the pathogenesis of BPH.. Androgens act through androgen receptors (ARs) in urogenital sinus mesenchyme (UGM) and this induces prostatic development. Homeostatic mechanisms between stroma and epithelium maintain the relative quiescence in adults. Androgens are involved in both the normal function of the prostate but also in prostate disease [41]

Though DHT is the primary growth hormone for prostate, it should be noted elevated DHT levels are not in association with human BPH.[42] The paradoxical observation of increasing prostatic growth despite declining androgen levels suggests that other factors secreted by the testis are responsible.[43] Estrogen in combination with other hormones may cause hyperplasia of the gland.. Estrogen via ERa, will causes aberrant cellular differentiation and proliferation with progression to prostatic hyperplasia and sometimes neoplasia, and dysplasia. [44,45,46]

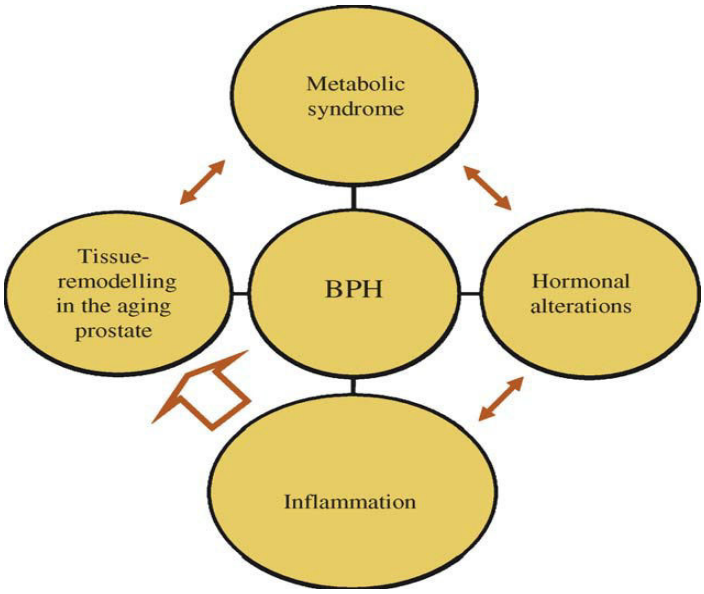

Figure 3
Pathogenesis of BPH

Cunha et al.[30,44] described that the modulatation of the differentiation pattern of prostatic epithelium is done by the stroma. Aberrant peptide factors are implicated too. However key cell is the " stromal cell" as it secretes many growth factors, which act in an autocrine manner on the stroma and neighbouring cells to cause increased growth.

There are qualitative and quantitative changes in the extracellular matrix. The role of glycosaminoglycans and Epidermal growth factor is under study..[47]

Infection may play a role in the pathogenesis of BPH.[48] Many reviews on the pathogenesis of BPH have suggested a role of inflammation in the propagation of histological BPH.[49,50,51,52,53] Kramer and Marberger[53] have given the current concepts of the role of inflammation. Chronically activated T cells and macrophages are associated with formation of BPH nodules. Production of cytokines (IL-2

and IFNγ) support fibromuscular growth. [53] Surrounding cells die, leaving empty spaces that are replaced by fibromuscular nodules with a specific pattern of a Th0/Th3 type of immune response.

There is increased evidence of a familial association as shown by Sands et al(1994) and Robert et al(1997)

Pathophysiology of BOO

There are various terms in use in order to try and describe and quantify the bladder and lower urinary symptoms secondary to prostate enlargement. As to if any one of them is singularly representative the answer is no. The partially obstructed urethra, detrusor muscle and the central nervous system function interact to produce lower urinary tract symptoms. These were historically referred to as 'prostatism'.

There are several mechanisms by which Benign prostatic hyperplasia (BPH) may cause obstruction such as

- A big median lobe acting as a ball valve
- A dynamic obstruction related to the contractile prostatic smooth muscle
- A static obstruction resulting from an enlarged prostate enveloping the prostatic urethra, or a restricted surgical capsule.

Each of these mechanisms is clinically feasible and components of each are likely to be present in most instances. The result is a raised intravesical pressure and a reduction in flow, which leads to the gradual development of secondary changes in the muscle itself.

Histologic Features.

Benign prostatic hypertrophy as a term is pathologically not correct.

It is a true hyperplasia as evidenced by histological studies[35] (McNeal, 1990).

McNeal's studies demonstrated that majority of early periurethral nodules were purely stromal in character[36].

During the early years of its development, BPH may be predominantly characterized by an increase in the number of nodules, and the growth of each new nodule is generally slow[35,36] (McNeal, 1990). Then a secondary phase occurs in which there is a growth of cells in large nodules.

There is pleomorphism in the ratio of stromal-epithelial tissues. Small glands demonstrate a more fibromuscularstroma[55](Shapiro et al, 1992b) whereas larger glands show epithelial nodules[56](Franks, 1976).

Importance of Prostatic Smooth Muscle

Prostatic smooth muscle forms a major portion of the enlarged gland.[55](Shapiro et al, 1992a). Both active and passive forces in and aroundprostatic tissue play a major role in the pathophysiology of BPH[56](Shapiro et al, 1992), leading to a mechanical and dynamic obstruction. α-Adrenergic blockade leads to a significant downregulation of smooth muscle myosin heavy chain leading to decrease in dynamic obstruction[57](Lin et al, 2001).

Effect of obstruction on the bladder:

Gross anatomical, histological, cellular and molecular alterations in bladder wall, which result from obstruction of the urethra impair its function and add to the symptomatology of BPH[59]. Detrusor muscle Hypertrophy compensates in early phases of outflow obstruction. With persistent obstruction however, decreased compliance in the detrusor and impaired emptying occur owing to the deposition of extracellular matrix (ECM)[60].Acute urinary retention may occur during the process and may be related to bladder failure, as well as to sudden increase in outflow obstruction. The alteration in ECM is probably the predominant

pathophysiological feature in long-term obstruction. Studies from the rabbit model of obstruction have shown that significant smooth muscle hyperplasiais induced when the load is increased and that this is associated with down regulation of myosin light chain (MLC) Expression. This effect contributes to the decreased smooth muscle contractility and moreover results in development of dedifferentiated smooth muscle phenotype[61].

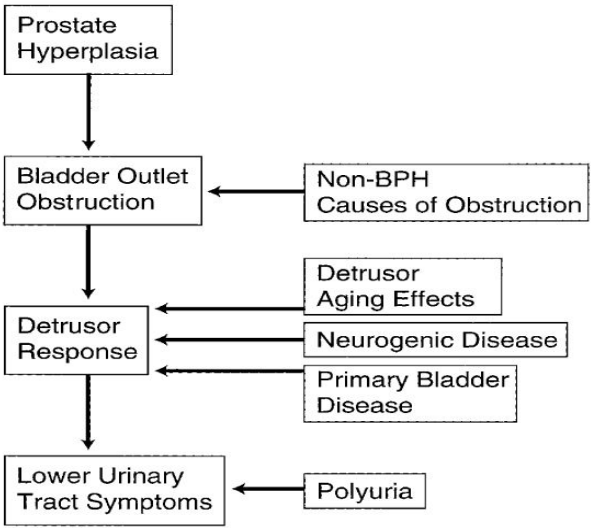

Figure 4:The pathophysiology of BOO9

Lower Urinary Tract Function of Ageing

CNS effects and changes in adjacent organ systems due to aging may increase LUTS [80].

The degree of the prostatic enlargement does not correlate with the symptoms.

Various drugs used for therapeutic purposes may induce LUTS in some individuals with a normal lower urinary tract[62].

Severe symptoms, bladder dysfunction, infection, CKD etc are the clinical endpoints. Olmstead County Study gave us insights regarding the natural progression of benign prostatic enlargement[62].

Lower Urinary Tract Symptoms

The nomenclature of voiding dysfunction in aging men is confusing and often inaccurate[67]. The term BPH should be used with reference to the histologic process of hyperplasia, which can be demonstrated microscopically. Men with benign prostatic enlargement (BPE) presumably have an increase in total prostate volume because of BPH. BPE may or may not produce clinically significant LUTS and may or may not produce urodynamically proven bladder outlet obstruction

Lower urinary tract symptoms (LUTS) is the current favored terminology for urinary symptoms [67]. According to the International Continence Society (ICS), LUTS means:

- a symptom, - this is perceived by the subject;
- a sign – which is observed by the physician;

- a condition – which is defined by urodynamic investigations [68]

Abrams et al classified the symptoms into three categories which was incorporated in the ICS system: 68

- storage symptoms - due to disturbances when urine is stored in the bladder
- voiding symptoms - problems when voiding.
- post micturition symptoms - immediately after micturition.

Prediction of Bladder Outlet Obstruction :

There are several tools which have been used to evaluate the degree of BOO due to BPH

- Symptom Scores / QoL
- Prostate volume
- Qmax
- Bladder outlet obstruction index / Bladder contractility Index
- Postvoid Residual urine
- PSA

All these parameters by themselves and its combinations were not very predictive of the need for surgery requirement in BPH patients. The era of medical management of the BPH futher muddled the picture. Can we

predict which patient can be managed successfully on medical management?

These questions brought forth a new set of non invasive Ultrasound parameters to help us assess the BOO

- Total prostate volume
- Transitional zone volume
- Transitional zone index
- Intravesical prostatic protrusion
- Resistive index
- Detrusor wall thickness
- Ultrasonic estimation of bladder weight.

Prostate volume

Larger the baseline size, greater is the progression as shown by Jacobsen and colleagues[63]. The AUA symptom score was classified as mild (0-7) or moderate to severe (8-35). The mean symptom score progression was maximally seen in the 60 to 69 year old age group. The Medical Therapy of Prostatic Symptoms study is the longest placebo-controlled trial follow up trial for BPH.

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Does the volume of prostate gland correlate with the degree of obstruction? Volume measurement can be achieved by atransrectally or

suprapubically done USG. Eventhough transabdominal USG is less than ideal, it correlates linearly with transrectal measurement.[59] Are large glands really more obstructive?Lepor et al concluded that total prostate and transition zone volumes were not correlating to symptom scores and only weakly with PFR[89]. The patients presenting with AUR had successful TWOC if the prostate size was smaller[90]. There is only indirect proposition that larger prostates have a tendency to be obstructive.

METHODS OF ESTIMATING THE PROSTATE SIZE

Size of the prostate can be estimated by transabdominal, transrectal, and transperineal ultrasonography. Since the specific gravity of the gland is nearly the same as that for water, volume is roughly equivalent to weight of prostate i.e. 1 cm^3 equals approximately1 gram of prostate tissue. For the prostate volume calculation the following dimension are required.

- Axial plane - anteroposterior dimension and the transverse dimensions.
- Sagittal plane - longitudinal dimension (measured just off the midline)

There are various methods for measuring the volume, serial

planimetry, three dimensional imaging and simple calculations from orthogonal ultrasound based measurements using geometric methods such as formula for sphere or prolate ellipsoid.

Most formulas were devised assuming that the gland conforms to an ideal geometric shape, i.e

- Ellipse= π /6 x TS x AP x CC,
- Sphere= π /6 x TS^3 ,
- Prolate (egg shape) / spheroid = π 6 x TS^2 x AP.

Despite the inherent inaccuracies that arise from these geometric assumption all the formulas reliably estimates gland volume, since the correlation coefficients between sonographic volume and radical prostatectomy specimen weights were found greater than 0.90.

The prolate ellipsoid method is simple and standard for calculation of prostate volume which uses the following formula:

Transverse diameter x Anteroposterior diameter x Length x 0.52.

The software programmed on most machines calculates the volume.

IMPORTANCE OF THE PROSTATE SIZE

Accurate prostate volume measurement is important for several reasons, when invasive surgery is considered the urologist opt for open surgery when the size of the prostate exceeds 75 cm^3 . Otherwise transurethral resection becomes the choice.Size selection is also considered in medical management. The treatment with 5 Alfa reductase inhibitors is less effective when the size is less than 50 cm^3.[59]

Prostate growth appears to be directly correlate with prostate gland size. The size of the gland was also to be an independent predictor of treatment intervention. Baltimore Longitudinal Study of Aging also showed that a large prostate gland is a good correlator with prostatectomy. Prostate Size Is a Surrogate for PSA. It gives us a idea regarding the size and also the rate of progression of disease.

Digital rectal examination is an inaccurate determination of prostate size and, in fact, appears to significantly underestimate prostate volume.Clinical trial data further confirm the utility of prostate volume in determining risk ofBPH progression. Strong evidence exists showing that baseline prostate volume andserum PSA level predicts future prostate growth.

IPP:

Kessler *et al*.[15]- Detrusor thickness > 2.9mm by ultrasound - high predictive value for BOO.

Tan and Foo[16]proposed the use of IPP as a parameter to predict the outcomes of voiding trial.

Chia *et al*.[19]concluded that IPP is a significant parameter to predict BOO – as good as urodyanamic parameters.

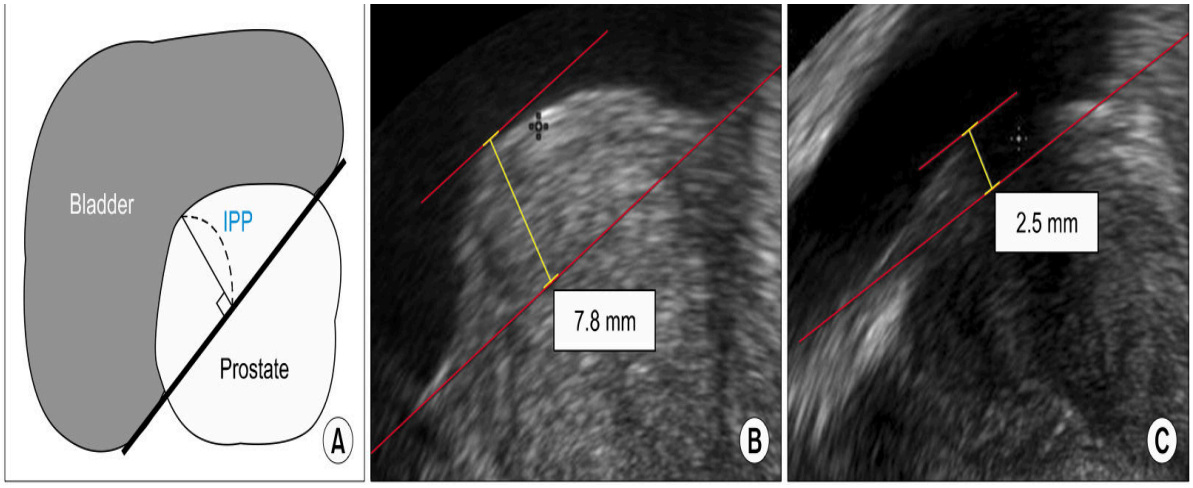


Figure 5: Measurement of IPP

Postvoid Residual Urine

Postvoid residual (PVR) urine is the volume of urine remaining in the bladder just after the completion of act of micturition. Studies indicate that PVR urine normally ranges from 0.09 to 2.24 ml (Hinman and Cox,

1967). 78% of normal men have PVRs of less than 5 ml, and 100% have volumes of less than 12 ml (Di Mare et al).

The AHCPR BPH Guideline Panel (Mc Connell et al, 1994) and Fourth International Consultation recommends that PVR is not a good parameter for diagnosing BOO due to its significant intraindividual variability and its poor correlation with signs and symptoms.

PVR measurement can be done by both noninvasive (ultrasound) and by invasive (catheterization) methods. However the most common method is by ultrasound. Invasive techniques are accurate but do carry a risk of discomfort, urethral injury, infection, and transient bacteremia

Prevalence of Lower Urinary Tract Symptoms

The overall prevalence of symptoms of the LUT varies tremendously in various reports [84,85,86,87,88]. One easy explanation is that different symptoms are included (Thom 1998). Each study had their own definition of symptom itself, as well as its frequency and severity. Another explanation for the large difference is the variety of population selected.

Assessment of Lower Urinary Tract Symptoms

For a subjective assessment of LUTS, several instruments have been developed. Frequency description is a first step toward understanding their impact [69].

The Boyarsky Score [70] is the first published questionnaire but never been validated. It evaluates the severity of nocturia, frequency, hesitancy, intermittency, terminal dribbling, urgency, reduction of the size and force of the stream, dysuria and incomplete voiding.

The Madsen-Iversen Score [71] is also not a validated questionnaire comprising of straining to void, hesitancy, intermittency, bladder emptying, stress incontinence or post void dribbling, urgency, frequency and nocturia.

The ICS male SF questionnaire allocates 11 questions on LUTS (hesitancy, straining, decreased stream, intermittency, incomplete emptying, urgency, urge incontinence, stress incontinence, unpredictable incontinence, nocturia, post-void dribbling) and one question on quality of life [72].

The Danish Prostatic Symptom Score (DAN-PSS) [74] measures the 12 symptoms of lower urinary tract measuring both quantitatively and qualitatively with the use of a symptom score and a distress score.[79].

The AUA symptom index is a validated questionnaire and includes seven questions covering frequency, nocturia, hesitancy,

intermittency, incomplete emptying, weak urinary stream and urgency and two questions on quality of life [69].

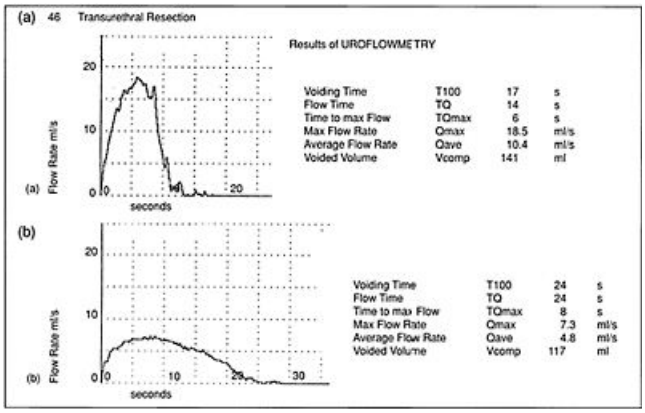
INTERNATIONAL PROSTATE SYMPTOM SCORE (IPSS)

The international Prostate Symptom Score (I-PSS) is based on AUA symptom index, which finds the answers to seven questions concerning urinary symptoms.[80]The answers are assigned points from 0 to 5 and so the total score can range from 0 to 35 (asymptomatic to very symptomatic). This is recommended for not only the initial evaluation but also during and after treatment for follow up.

Uroflowmetry

Uroflowmetry is a easy, office screening procedure where we measure the rate of flow of urine over time. The test is noninvasive and may be used to evaluate bladder and a little of sphincter function. The information guides us to functionally evaluate the lower urinary tract and to determine whether there is an obstruction of normal urine outflow.

It is a common, noninvasive urodynamic test used in the evaluation of patients presenting with symptoms of BOO.



Figur6 :Uroflowmetry Curve

The AHCPR Guideline Panel (McConnell et al, 1994) and Fourth International Consultation on BPH came out with various recommendations regarding Uroflowmetry. They concluded that flow rate – peak and average maybe the single best noninvasive test to predict BOO. However a cutoff value is difficult to predict. It also fails to differentiate a obstructed bladder from a decompensated bladder.

Inspite of this limitation, flow rate showed some sensitivity in diagnosing the degree of BOO due to BPH. (Scott et al(1967); Shoukry et al (1975); Siroky et al (1979) Gleason et al (1982).

However Chancellor and colleagues (1991) demonstrated that flow rate cannot distinguish between BOO and inadequate detrusor contractility as the cause for BOO. Abrams and associates (1997) found low failure rates for surgery with the addition of PFR measurement to symptom assessment in preoperative evaluation. This was also proposed by Jensen et al in 1984.

McLoughlin and coworkers, in 1990, using a cutoff value of 12 mL/s, proved that only 3% of patients would have undergone an unnecessary TURP. However very low rates do not appear to portend poor treatment outcome.

Both subjectively assessed symptom and quantified symptom score analysis donot correlate with uroflowmetry measurements; they are independent assessments.

Currently impaired compliance remains the only "absolute urodynamic indication" for treating BOO.

HISTORY OF PROSTATE SURGERY HISTORICAL REVIEW

The term prostate is derived from the writings of Herophilus (300 BC), of Alexandria who performed cadaver dissection.

The word "Prostate" means "stand before" i.e. it stands at the exit of the bladder.

1) Oribasius (325 – 403 A.D) was the first to describe the prostate gland as a swelling at the bladder neck.

2) Massa, a Venetian physician, published detailed description of the gland in 1536.

3) The oldest illustrations of prostate are found in

“TabulaeAnatomicae” published by Vesalius in 1538.

4) The role of the prostate in bladder outlet obstruction has only been recently understood. Prostatic hypertrophy was unknown in ancient Egypt (Rowling 1967) and there is no reference to prostate in Hippocratic writings.

5) As life expectancy was short, prostatic hypertrophy was probably uncommon, but descriptions of urinary retentions were not rare and the ancient Indians and Chinese practiced catheterization. Retention was frequently attributed to bladder stones or venereal strictures.

6) Pare described prostatism in 1564, but attributed outflow obstruction to callosities of the bladder neck or to feeble expulsive efforts in the elderly.

7) In 1649, Riolan first recognized outflow obstruction caused by prostatic enlargement.

8) In 1762 Morgagni confirmed these findings.

9) In 1788, John Hunter described middle lobe and lateral lobe enlargement and the effects of prostatic hyperplasia on the bladder and upper urinary tracts.

10) The term “hypertrophy” was coined by Mercier in 1841, who also

explained the basis of retention with overflow.

11) In 1902, Albarran and Motz established the role of periurethral glands in the development of prostatic hyperplasia.

EARLY FORMS OF TREATMENT:

1) For thousands of years, catheterization remained the cornerstone of treatment for retention. Catheters are described in the annals of ancient India, China and Greece.

2) Catheters were usually made of bronze, but other metals and wood or reeds were also used.

3) Oribasius developed indwelling catheters made from treated paper.

4) In the middle age, wax catheters were developed.

5) The credit for developing gum elastic catheters goes to Bernard, a Paris goldsmith and surgical instrument maker.

6) In 1836, Merck developed his coude catheter and in 1841, the bicoude catheter to negotiate difficult strictures.

7) Reyband designed the first self retaining balloon catheter around the same time, a forerunner of the Foley catheter, that revolutionized indwelling catheterization.

8) Catheterization remained the mainstay of treatment for obstruction until less than 100 years ago.

9) Alternative methods, with varying degrees of success included the administration of hemlock and ergot, prostatic massage, and intraprostaticsclerotherapy.

10)Dilatation of the prostatic urethra was practiced by John Hunter and by Harrison (1881) who used graduated dilators.

11) Physick of Philadelphia used a bag attached to a catheter which was inflated and left for 15 minutes.

12)Vasectomy and orchidectomy were done based on Hunter's (1786) observation that in young male animals, the prostate failed to grow following castration.

13) Albarran and Motz in 1898 demonstrated that these methods were ineffective except in cases of prostatic carcinoma.

14)Perineal lithotomy has been practiced since early times. Conville in 1639 crushed a firm (prostatic) tumour before removing a bladder stone and Desault in 1791, twisted off a prostatic tumour during lithotomy, relieving the patient of obstruction.

15) Ferguson in 1870 also reported similar procedure.

16)Goodfellow is credited with performing the first perineal prostatectomy in 1891 and reported a series of 78 cases with only two deaths in 1904.

17) Prostatectomy through the extra urethral perineal approach was probably first performed by Billroth in 1867.

18) This approach was further developed by Langenbeck and by Zukerkandl.

19)Complete perineal prostatic enucleation was developed independently by Proust (1901) and Hugh Hampton Young (1903). Young's operation has remained the standard perineal approach.

RECENT HISTORY

TURP, as we know it today, was developed in the United States in the 1920s and 1930s. Nesbit (1975) pointed out that there were several significant factors important in its development

1) The invention of the incandescent lamp by Edison in 1879.

2) The cystoscope, developed independently by Nitze and Lieter in 1887,

3) The development of the fenestrated tube by Hugh Hampton Young, which allowed the obstructing tissue to be sheared off blindly.

MATERIALS AND METHODS

1. Study group:

Patients who were admitted in Kilpauk Medical College and Govt. Royapettah Hospital with lower urinary tract symptoms (LUTS) due to benign prostatic hyperplasia (BPH) was included in the study.

2.Study design: Prospective observational study

3.Materials :

Estimated prostate size of 30gm or more, which was determined by digital rectal examination (DRE) and ultrasonography using prolate ellipsoid formula

(Prostate size in gm= $\pi/6$ ×anteroposterior × transverse × sagittal diameter).

These patients were evaluated for IVPP by transabdominal ultrasound (Phillips 3.5MHz curvilinear probe). The bladder should have a capacity of 150–200 mL before the extent of IPP was measured. IPP is assessed by measuring the vertical distance from the tip of the protrusion to the bladder wall at the base of the prostate gland.

They were grouped into three groups based on it

Group A – IVPP <5 mm

Group B – IVPP 5 - 10 mm

Group C – IVPP > 10 mm

4. Study period – 1 year

Inclusion criteria –

Patients undergoing TURP for the following reasons;

1. Prostate size> 30gms
2. AUR
3. Maximum flow rate (Qmax) < 15 ml/s
4. Postvoid residual urine (PVR) > 100 ml
5. Complications like bladder stones and hydroureteronephrosis.

Exclusion criteria –

- Prostate cancer
- PSA ≥4 ng/ml
- Stricture disease

- Neurogenic bladder
- Previous surgeries of prostate or urethra
- All cases which do not come under inclusion criteria
- Unwilling patients

Preoperative variables (symptom scores, QoL, PVR, uroflowmetry parameters) comparable in between the two groups

Intraoperative variables (operating time, amount of irrigation fluid and blood transfusion required) was observed and recorded.

Postoperative catheterization period and hospital stay (in days) were noted.

Post operative variables (symptom scores, QOL, PVR, uroflowmetry parameters) were be compared.

OBSERVATION AND RESULTS

We evaluated the impact of the degree of IPP on the outcome of TURP in our institution.

In our study, total of 60 patients over a period of one year were included according to the required criteria. They were preoperatively evaluated for the extent of IPP and were divided into three groups according to the IPP.

Group a – IPP <5 mm

Group b – IPP 5 - 10 mm

Group c – IPP > 10 mm

SPSS version 16.0.0 (SPSS Inc, Chicago, IL, USA) was used for the statistical analysis

Table 1 :AGE DEMOGRAPHY

			GROUP			
			a	b	c	Total
age	(<60 YRS)	Count	10	4	1	15
		% within GROUP	50.0%	20.0%	5.0%	25.0%
	>60 YEARS	Count	10	16	19	45
		% within GROUP	50.0%	80.0%	95.0%	75.0%
Total		Count	20	20	20	60
		% within GROUP	100.0%	100.0%	100.0%	100.0%

Age:

Age ranged from 50 – 81 years. Mean age in all groups was 65 years. It was found that as group b & c had more > 60 years old. This may indicate the correlation of IPP with age.

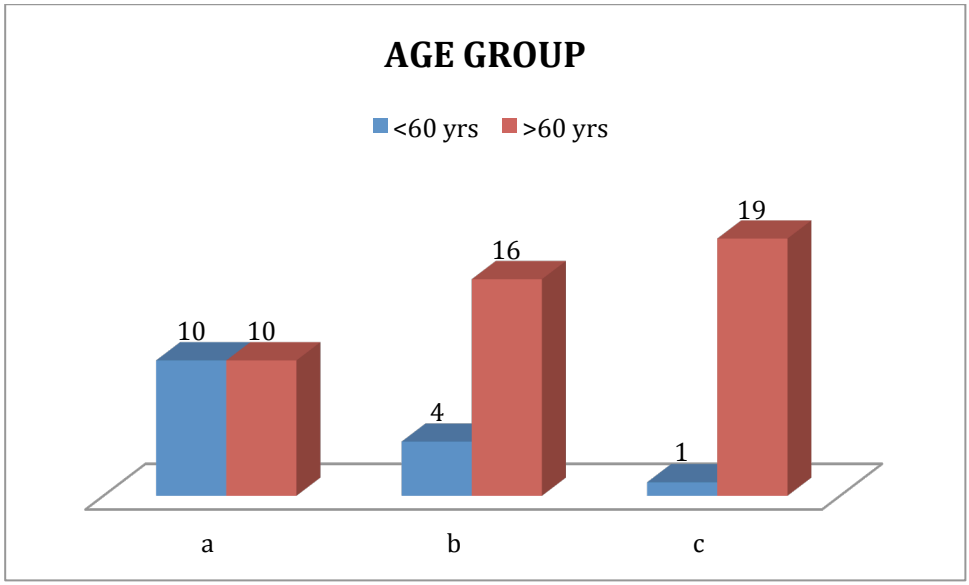


Table 2: GLAND SIZE AND PSA (MEAN AND SD IN EACH GROUP)

GROUP		PSA	GLANDSIZEGMS	p
a	Mean	2.005000	53.45	0.14
	Std. Deviation	.9098727	8.888	
b	Mean	3.305000	57.65	
	Std. Deviation	5.4091030	8.331	
c	Mean	1.670000	61.20	
	Std. Deviation	.8367229	6.779	
	Mean	2.326667	57.43	

There was no statistically significant difference betweenthe groups with respect to the mean values of PSA and gland size.

Table 3:IVPP BETWEEN THE GROUPS

GROUPS	Mean±SD	P
a	4.05±2.32	
b	7.63±1.37	0.000
c	16.37±1.32	

The mean IVPP was compared between the three groups using one way ANOVA, and it was found to be highly significant (p= 0.000)

Table 4: HOSPITAL STAY, IRRIGATION FLUID AND OPERATIVE TIME

GROUP		OPERATIVETIME	IRRIGATION FLUID	HOSPITALSTAY
a	Mean	58.20	13.35	4.80
	Std. Deviation	7.838	2.758	.696
b	Mean	56.80	14.00	4.80
	Std. Deviation	7.466	2.492	.696
c	Mean	55.95	14.85	4.85
	Std. Deviation	7.200	2.498	.366
P value		0.634	0.163	0.956

There was no difference with respect to the operative time, hospital stay and amount of irrigation fluid used

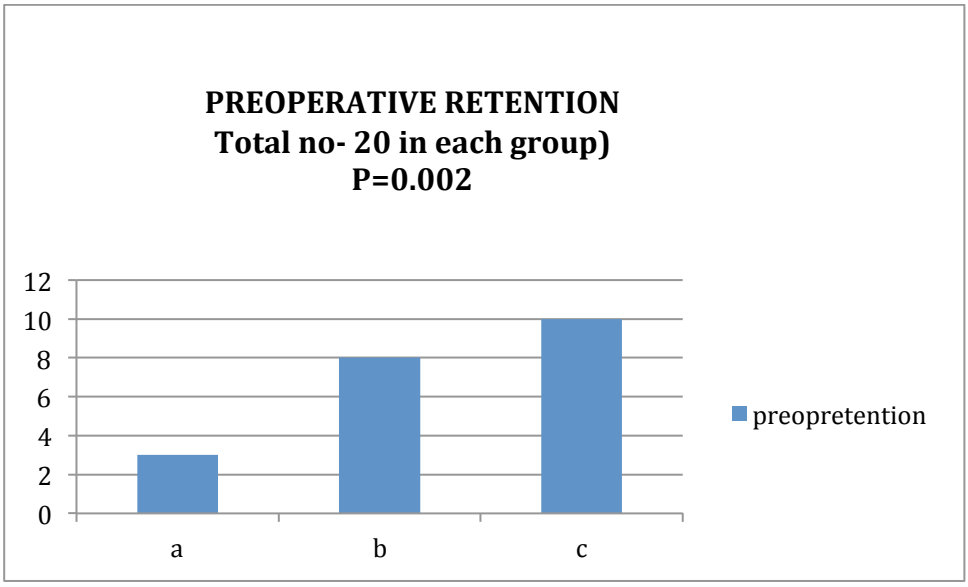
Table 5: PRE OPERATIVE RETENTION

Group	Number (%) N=20 in each group	p
a	3(15%)	
b	8(40%)	
c	10(50%)	0.002

The pre operative retention was compared between the three groups using Chi Square test and it was high in Group C when compared to b and a (p=0.002).

Table 6: COMPARISON OF IVPP WITH PSA, PREOP PVR , PREOP PFR AND PRE OP RETENTION

IVPP / GROUP	PSA (p)	PRE OP PFR (p)	PRE OP PVR (p)	PRE OP RETENTION (p)
	0.918	**0.487**	**0.019**	**0.003**

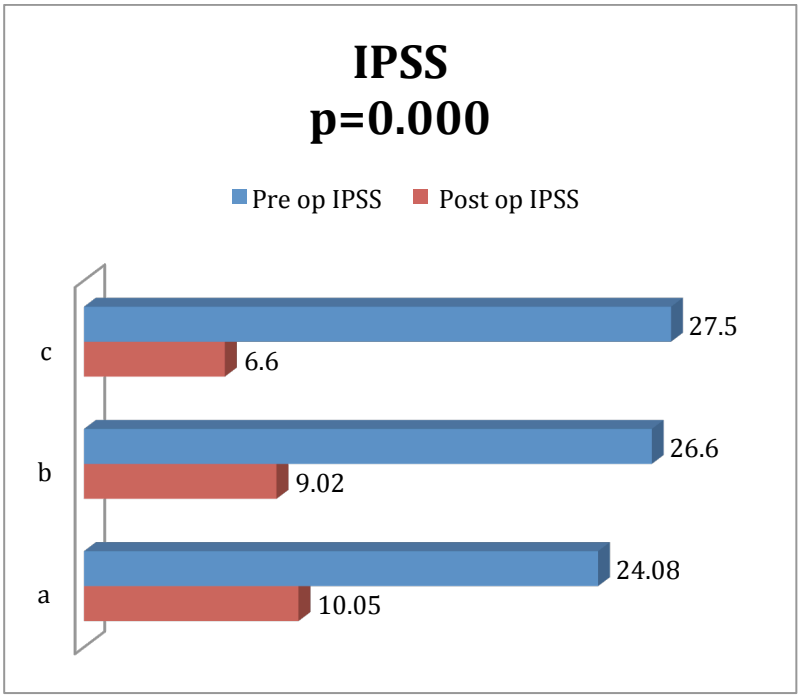


The IVPP was compared with the PSA values, pre operative PFA and PVR values using One way Anova TEST. The values of the same when compared to IVPP was not significantly different in either of the groups.

We compared IVPP with the pre op retention using chi square test which was found to be statistically significant

Table 7: IPSS

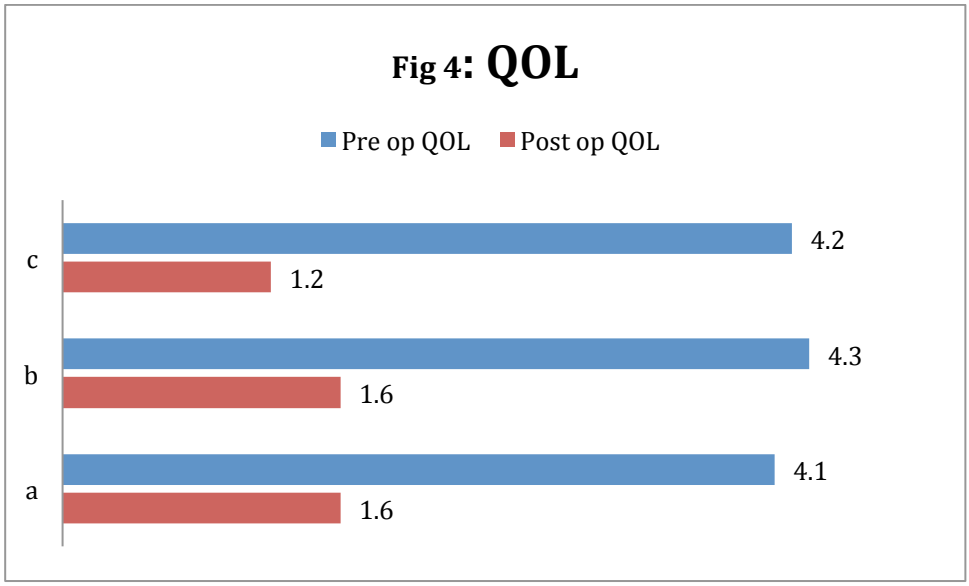
GROUP	Pre op IPSS	Post op IPSS
a	24.08±3.1	10.05±2.5
b	26.6±2.8	9.02±3.4
c	27.5±4.5	6.6±1.3
P value	0.058	0.000



The pre op and post op IPSS were compared between the groups. Though the pre op IPSS was almost the same in the three groups, the post op IPSS was significantly lower in Group C. (p=0.000)

Table 8: QOL

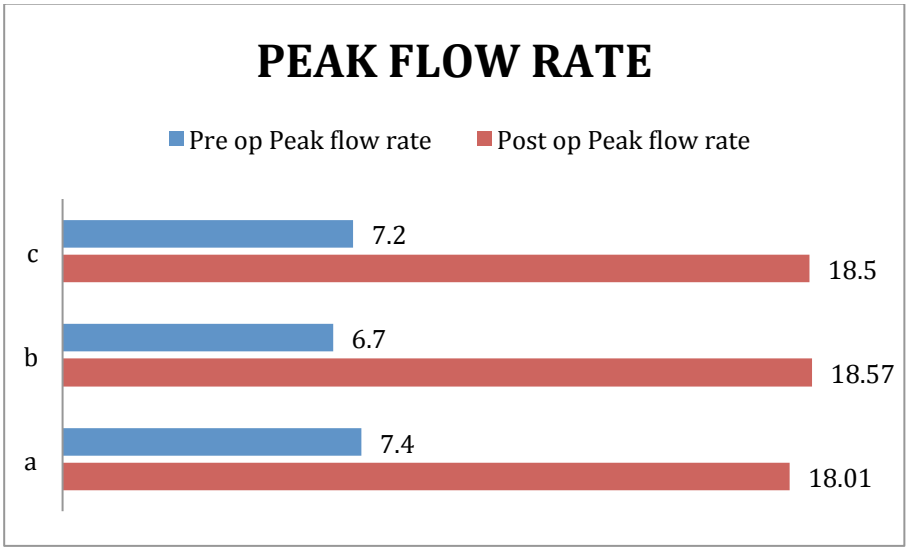
GROUP	Pre op QOL	Post op QOL
a	4.1±0.3	1.6±0.5
b	4.3±0.4	1.6±0.6
c	4.2±0.4	1.2±0.4
P value	0.09	0.047



The QOL before and after surgery was compared between the groups. There was a clinically significant higher QOL in Group C when compared to a and b.

Table 9: PEAK FLOW RATE

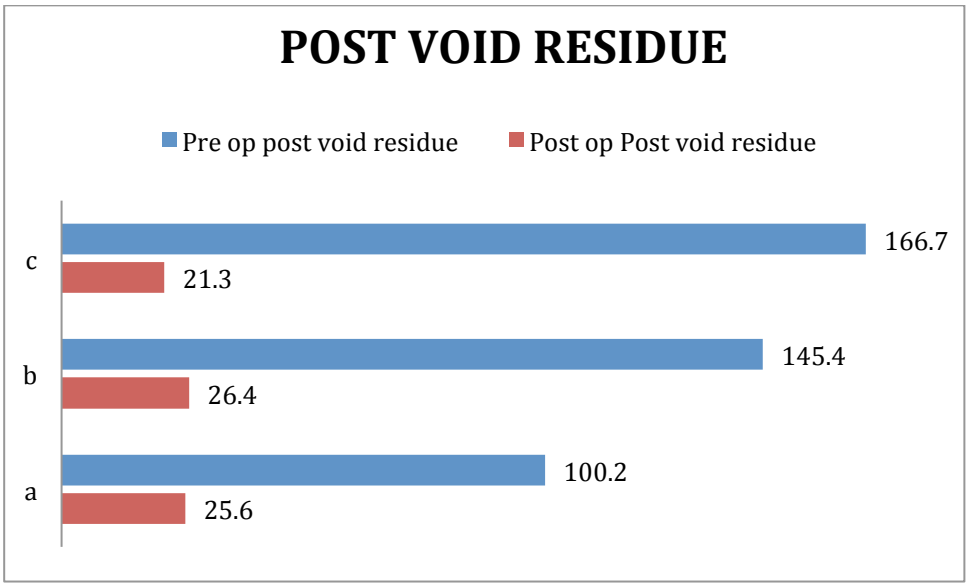
GROUP	Pre op Peak flow rate	Post op Peak flow rate
a	7.4±1.5	18.01±2.32
b	6.7±1.2	18.57±2.51
c	7.2±1.9	18.5±2.43
P value	0.508	0.49



There was no significant difference in the means of the pre operative and post operative PFR between the groups

TABLE 10: POST VOID RESIDUE

GROUP	Pre op post void residue	Post op Post void residue
a	100.2±17.09	25.6±9.8
b	145.4±88.3	26.4±2.10.9
c	166.7±103.5	21.3±31.7
P value	0.06	0.83

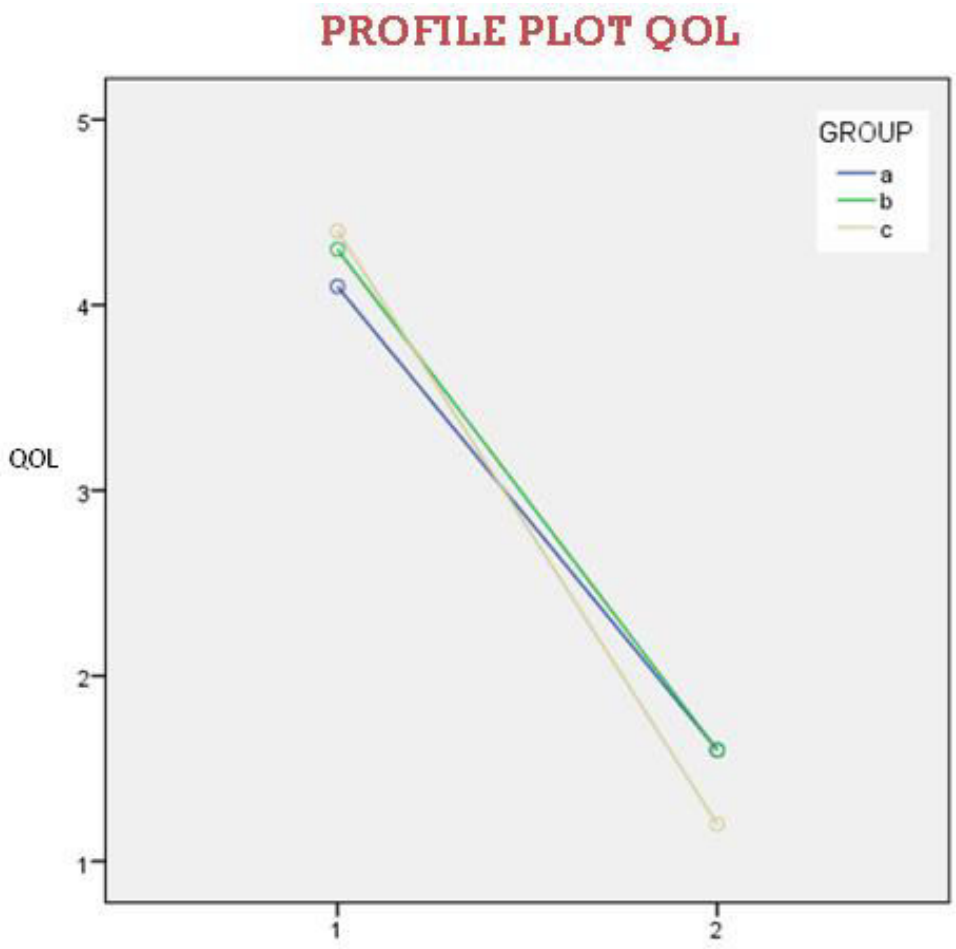


There was no significant difference in the means of the pre op and post op PVR between the groups. But many patients in Group c were catheterized and hence their PVR could not be measured.

In order the compare the **change** in the QOL, IPSS, PVR and PFR between the three groups, a repeated measures ANOVA test was performed and the profile plots of the change in each of the parameters was plotted. There was a significant clinical betterment in terms of QOL and IPSS in Group c when compared to the other 2 groups.

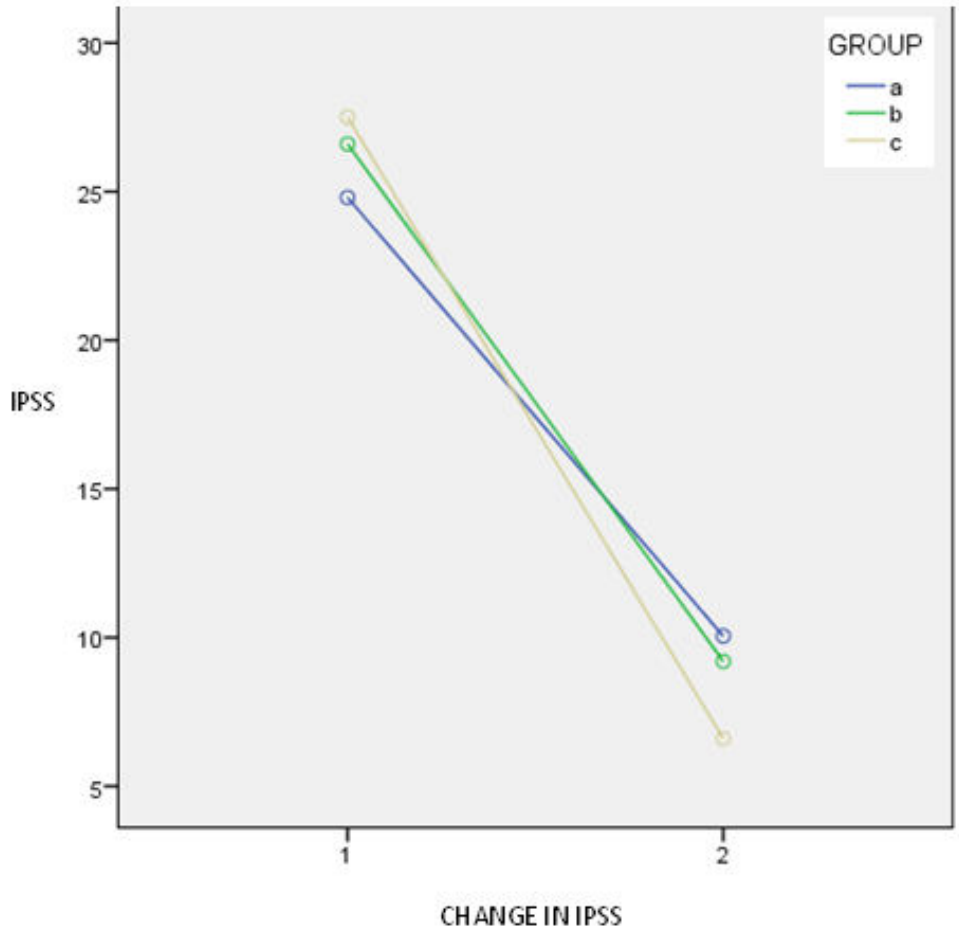
Similarly the profile plots of the change in theUroflometry variables (PFR and PVR) again showed a clinically significant improvement in Group c when compared to a and b

Using Repeated measures ANOVA test, the four variables IPSS, QOL, PFR and PVR were compared between all the groups and the profile plots were plotted.

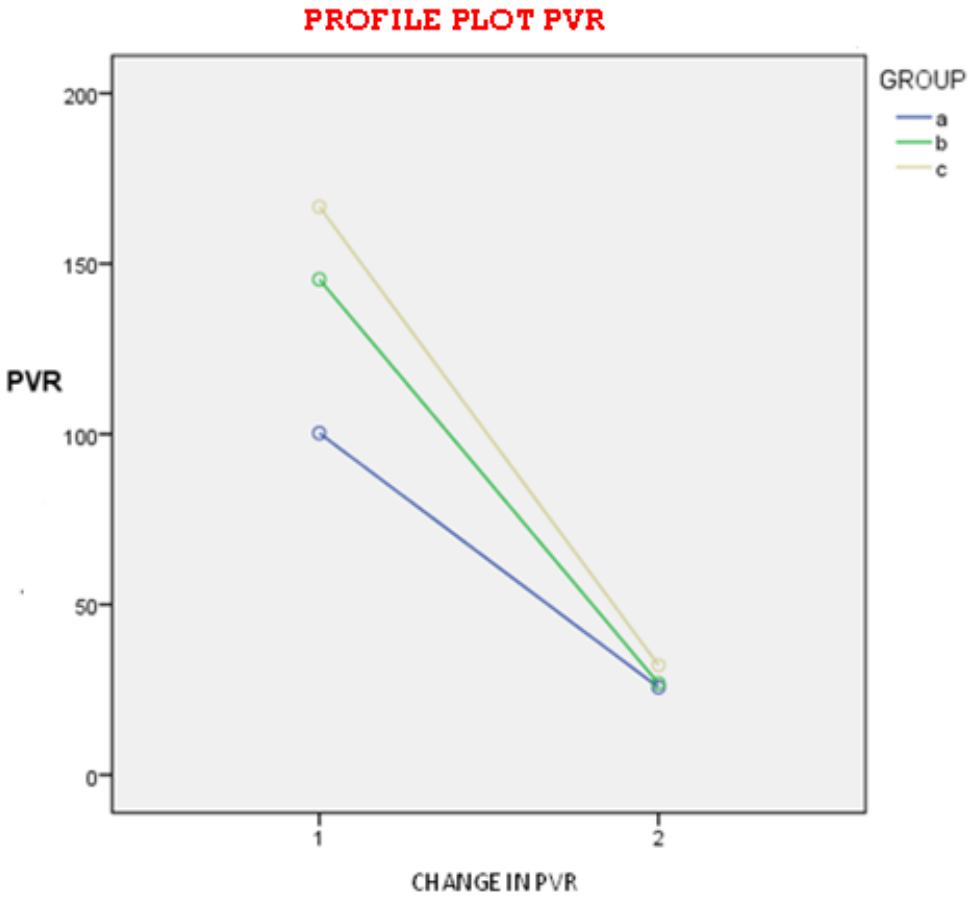


The p value for the change of QOL was clinically significant (p=0.47). Group c had a greater betterment in QOL when compared to a and b

PROFILE PLOT IPSS

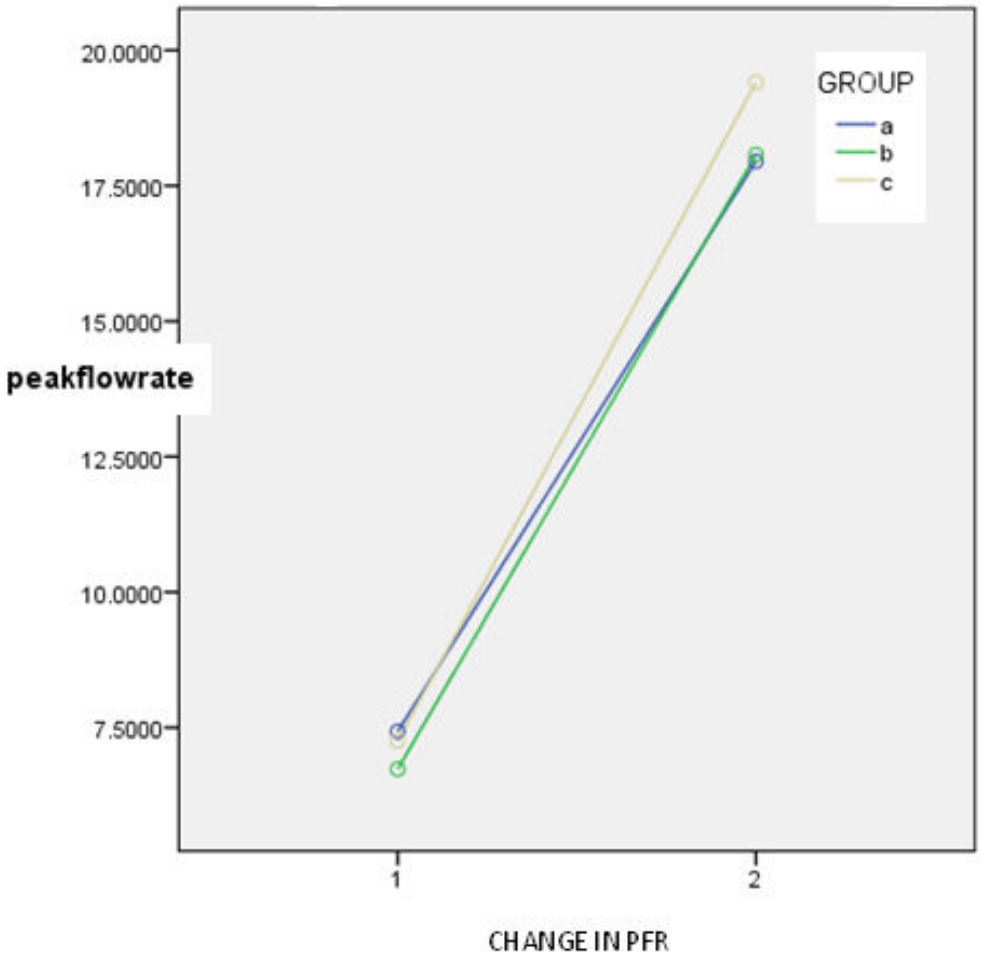


The change in IPSS was significant between the groups (p = 0.542)


PROFILE PLOT PVR
GROUP
a
b
c
200
150
100
50
0
PVR
1
2
CHANGE IN PVR

p= 0.106

PROFILE PLOT PEAK FLOW RATE



p=0.46

DISCUSSION

Till date pressure flow study parameters remain the gold standard for assessing bladder outlet obstruction. Urodynamics is expensive and needs expertise and is invasive.

Intravesical prostatic protrusion (IPP) is defined as the degree of protrusion of the prostate from the base of the bladder into it. Several studies have shown this parameter to correlate well with predicting BOO in patients with BPH [21,22]. IPP also gives an indirect idea regarding the severity of BOO. This was exemplified by the paper by Franco et al. who demonstrated a good correlation between IPP and BOOI (Spearman's rho=0.49, p=0.001), and Schaefer obstruction class (Spearman's rho=0.51, p=0.001)[24].

Chia et al., showed that the higher the grade of IPP, the worser the bladder obstruction in their cohort of 200 patients, again reiterating the benefit of measuring the IVPP[19].

IPP can be measured by either transrectal or transabdominal USG. Suprapubic ultrasound is a simple, non-invasive and accurate method of assessing prostatic protrusion leading to bladder outlet obstruction in patients with BPE. TRUS is more invasive and painful to many patients. Painful anal conditions are a contraindication to TRUS and may not be

possible in all patients. Kim et al found no significant difference in prostate volumes by a transabdominal and transrectal ultrasound between observers in their study.

The purpose of our study is to assess whether the degree of IPP can successfully predict the outcome of a surgical intervention i.e. TURP. There is currently a need for a noninvasive parameter/s to successfully predict the outcome for both medical and surgical management of BPH. Is IPP the one noninvasive parameter that can point towards BOO?

The age distribution showed a higher percentage of patients above 60 years in Group b and c. This could be due to the increase in prostatic size with age. However there is no study till date, which correlates age with IPP. It was found in our study that most of our patients in-group c belonged to > 60 years. Prostate growth appears to be related to prostate volume. In the Olmsted County population-based study, men who had baseline prostate volumes of 30 mL or less had median prostate growth of 1.7% per year, compared with 2.2% per year for men with prostate volumes greater than 30 mL. However the dogmatic view is that BOO symptomology has poor correlation with the size of the prostate.

The mean values of PSA and the prostate gland size were comparable between the groups. Only prostate size > 40cc were included in this study. This meant that the prostatic volume mean of all the three groups were comparable in spite of varying IPP sizes. This was indirect evidence that the factors affecting the outcome of the study were rather of IPP than the prostate volume.

The intravesical protrusion of the prostate may lead to a ball-valve type of obstruction effect disrupting the funneling effect of the bladder neck to increase urethral resistance and causes dyskinetic movement of the bladder during voiding.

The mean IPP in group a,b,c were 4.05, 7.63, 16.37 respectively.

The mean IVPP between the groups was highly significant (p=0.000). We compared the relationship ofIVPP with the level of obstruction pre operatively. The pre op retention was significantly higher in Group c (p=0.002). The number of patients in group c who had AUR were more (50%) than b (40%) and a (15%).

This is in accordance with the various trials involving IPP as a parameter. Lee et al. [18] considered the IPP as a probable predictor worsening clinical symptoms in men with BPH who were put on medical management. Sharis[26] etal proposed that IPP is an important predictor of

success of decatheterisation trial in patients with AUR. Grade 3 IPP (>10 mm) was found to be a significant factor in predicting the rate of successful trial without catheter. The difference was found to be quite significant when an intergrade comparison was done in patientswith failed Trial without catheter.

These findings were similar to the study by K.B. Lim et al[21] in Asian population and by Leonardo O Reis et al[25]in Latin American patients.

There was no statistical difference with respect to the operative time, hospital stay and amount of irrigation fluid used

The IVPP was compared with the PSA values, pre operative PFR and PVR values using One way Anova TEST. The values of the same when compared to IVPP was not significantly different in either of the groups. There are several reasons for the outcome of this comparison the most important of which is the small sample size.

The pre op and post op IPSS were compared between the groups. Though the pre op IPSS was almost the same in the three groups, the post op IPSS was significantly lower in Group c (p=0.000). The comparable Pre op IPSS between the groups may be because of the detrusor compensatory hypertrophy to overcome the obstruction. Due to the

subjective nature of the IPSS, patients on urinary catheter might have given a falsely low score

The QOL before and after surgery was compared between the groups. Like IPSS there was a clinically significant higher QOL in Group c when compared to a and b, again reflecting the greater betterment in the symptoms in-group c when compared to b and a.

There was no significant difference in the pre and postoperativeuroflowmetry variables(PFR and PVR) in the groups. But many patients in Group c were catheterized and hence their PVR could not be measured.

In order the compare the change in the QOL, IPSS, PVR and PFR between the three groups, a repeated measures ANOVA test was performed and the profile plots of the change in each of the parameters was plotted. There was a significant clinical betterment in terms of QOL and IPSS in Group c when compared to the other two groups.

Similarly the profile plots of the change in the Uroflometry variables (PFR and PVR) again showed a clinically significant improvement in Group c when compared to a and b. Jung et al, in their study, demonstrated that IPP is not a good predictor of a better surgical outcome of Qmax and PVR in their study group as in ours. They also documented that significant IPP was more associated with the symptoms

score improvement rather than the uroflow variables as found by the multivariate analysis. They were able to show a close connection between significant IPP and improvement in the IPSS and IPSS-s as it is with our group c. They proposed that the significant relationship between IPP > 10mm and decreased IPSS may be because of irritation of the bladder neck and trigone by IPP thereby worsening the storage symptoms.

Zhang Keqin et al[27]divided the patients into two groups - the significant IPP group (> 10 mm) and the non-significant IPP group (≤ 10 mm). He showed significantly higher PV, PVR ($P<0.05$) and IPP ($P<0.001$) in obstructed patients. But in the present study there was no significant difference in the Qmax. This may be due to secondary hypertrophy of detrusor from BOO.These results are comparable to the study conducted by Lim KB et al[21].

K.T. Foo et al[22]showed that Prostatic volume & IPP had excellent correlation with BOOI. His results are comparable to the results of this present study.

Not all patients who underwent TURP for BPH obtain results that are personally satisfactory to him and to the surgeon. If the postoperative outcome could be predicted beforehand, it would be very useful in planning the management. It is known that some factors such as size of

the prostate and high BOOI can predict a satisfactory outcome [6]. We propose that in addition to traditional preoperative parameters, IPP is a definitive important tool to predict a better surgical outcome.

To conclude IPP may well be considered as an important parameter for predicting postoperative outcomes in BPH patients who undergo TURP.

CONCLUSION

- Pressure flow study parameters remain the gold standard for assessing bladder outlet obstruction.Pressure flow studies are invasive and expensive.
- It is found that higher the grade of IPP, the worser the bladder obstruction.
- IPP grade is probably associated with worsening clinical symptoms in men with BPH who were put on medical management.
- There was no statistical difference with respect to the operative time, PSA, hospital stay and amount of irrigation fluid used.
- There was not much difference in improvement of PFR, PVR between all the three groups.
- The profile plots of the change in the Uroflometry variables (PFR and PVR) again showed a clinically significant improvement in Group c when compared to a and b.
- The post op IPSS was significantly lower in cases with IPP > 10mm when compared to the two groups with significant IPSS score difference.

- A clinically significant higher QOL in Group c (IPP>10mm) when compared to a and b, again reflecting the greater betterment in the symptoms
- IPP may well be considered as an important parameter for predicting postoperative outcomes in BPH patients who undergo TURP.

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INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE, CHENNAI-10

Ref.No.3393/ME-1/Ethics/2013 Dt:27.09.2013

CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval"A Study on relationship between intravesical prostatic protrusion and postoperative outcomes in patients with benign prostatic hyperplasia" – For Project Work. Submitted by Dr.Srivathsan.R, MCh. (Uro), PG Student, KMC, Chennai.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

GOVT. KILPAUK MEDICAL COLLEGE
25 NOV 2013
Kilpauk, Chennai-10.

CHAIRMAN, 25/11/13
Ethical Committee
Govt.Kilpauk Medical College,
Chennai

PROFORMA

NAME: AGE: SEX:

ADDRESS: IP.NO:

D.O.A: D.O.S: D.O.D:

PRESENTING COMPLAINTS:

H/O AUR

IPSS SCORE:

GENERAL EXAMINATION: P.R: B.P:

PER ABDOMEN:

CATHETER:

PER RECTAL:

INVESTIGATIONS:

HB%: PCV%

BLOOD: UREA- SUGAR-

SERUM CREATININE- ELECTROLYTES-

SERUM PSA-

URINE C/S:

USG KUB:

PROSTATE SIZE:

IVPP:

UROFLOW: Qmax:

AFR:

Voided Volume:

CYSTOSCOPY:

Presence of lateral and median lobes – Grade

Presence of intravesical extension

Length of prostatic urethra

OPERATIVE PROCEDURE:

Operative time: Irrigation:

POST OP.PERIOD:

CATHETER REMOVAL:

USG:

FOLLOW UP:

IPSS SCORE:

UROFLOW: Qmax

AFR

Voided Volume

BIOPSY:

IMPRESSION:

ANNEXURE -3

GROUP	NAME	AGEYEAR	IP NO	INSTITUTION	GLANDSIZE	PSA	IVPP	PREOPIPSS	POSTOPIPSS
a	mr.nateshan	54	11760	grh	66	3.1	5	28	10
a	mr.chelladurai	55	11520	grh	55	2.1	4	22	9
a	mr.elumalai	56	2435	kmc	43	2.4	2.5	18	11
a	mr.alaudhin	57	2345	kmc	54	1.2	3	24	10
a	mr ponnusamy	57	854	grh	38	1.2	4	21	14
a	mr.mari	58	11235	grh	45	3	5	22	10
a	mr yaseem	59	11324	grh	61	2.5	3	29	14
a	mr.venkatasamy	60	11567	grh	46	1.4	4.5	28	10
a	mr.elumalai	60	2540	kmc	57	0.8	3	28	14
a	mr.durai	60	3210	kmc	45	0.7	2	26	7
a	mr.abdhul samad	68	23546	grh	70	2.9	13	26	12
a	mr.muthusamy	69	21346	grh	45	3.1	3	24	6
a	mr.chandran	70	1124	kmc	62	2.7	4.5	23	10
a	mr.thulasi	70	28761	grh	50	1.3	5	30	9
a	mr.elumalai	70	1265	grh	62	1.8	2.3	23	10
a	mr.renganathan	70	2134	kmc	55	0.7	3	27	9
a	mr.arumugam	70	2345	kmc	50	2.6	4.6	23	7
a	mr.arunachalam	70	367	kmc	65	3.2	3.5	27	14
a	mr.manivel	71	22398	grh	45	2.6	3.8	25	8
a	mr.rayappan	71	2347	kmc	55	0.8	2.2	22	7
b	mr.perumal	60	3115	kmc	56	0.6	7.4	27	11
b	mr.vivekanandhan	60	3214	kmc	65	2.8	6	23	8
b	mr.rajagopal	62	11643	grh	72	1.5	9	27	9
b	mr ramasamy	63	25284	grh	68	1.9	8	24	7
b	mr.govindasamy	63	3453	kmc	63	3	6.5	25	8
b	mr.velu	63	3325	kmc	66	2.3	7	23	8

GROUP	NAME	AGEYEAR	IP NO	INSTITUTION	GLANDSIZE	PSA	IVPP	PREOPIPSS	POSTOPIPSS
b	mr.dharmar	64	24321	grh	54	1.9	9	26	10
b	mr perumal	64	23579	grh	48	2.4	6	20	8
b	mr.sambasivam	64	2341	kmc	55	1.4	8.5	26	5
b	mr.thirumal	65	24328	grh	54	2.5	9	27	7
b	mr.govindarajulu	71	2456	kmc	50	2.3	6	24	7
b	mr.arunachalam	72	2476	kmc	54	3.2	8.4	27	15
b	mr.appasamy	75	3487	kmc	65	4	9.2	28	7
b	mr.shanmugavel	75	27890	grh	46	1.4	6	28	6
b	mr.thanavel	80	3267	kmc	52	26	8.5	30	16
b	mr.mohamed sherif	81	26790	grh	60	3	6.4	29	9
b	mr.kannappan	60	2567	kmc	48	2.1	9.1	31	6
b	mr.kasi	60	3120	kmc	70	1.9	5.9	31	18
b	mr.ramanathan	69	2489	grh	45	0.7	6.8	27	11
b	mr.immanuvel	73	24768	grh	62	1.2	10	29	8
c	mr.krishnan	65	2871	kmc	60	2.1	12	27	6
c	mr.noorulah	65	22134	grh	64	3.4	20	21	5
c	mr.ganeshan	65	24357	grh	68	1.9	13.5	24	6
c	mr.chinnappan	65	2345	kmc	59	0.5	18	23	6
c	mr.pandurangan	65	23476	grh	72	1.1	19	24	7
c	mr.kaliyamoorthi	66	3215	kmc	64	0.6	16	29	8
c	mr.nagappan	67	24545	grh	70	1.3	14.5	21	5
c	mr.elumalai	67	2135	kmc	68	2.5	19	21	7
c	mr.sampath	68	3247	kmc	64	2.4	13	23	6
c	mr.subbiah	68	28712	grh	65	1.5	16.5	24	8
c	mr.thiruvenkatam	74	2340	kmc	50	0.9	13.5	30	9
c	mr.thankaraj	79	3217	grh	62	3.1	14	29	7
c	mr.ramukutty	80	2476	grh	55	2.8	18	31	9

GROUP	NAME	AGEYEAR	IP NO	INSTITUTION	GLANDSIZE	PSA	IVPP	PREOPIPSS	POSTOPIPSS
c	mr ganesan	70	21234	grh	50	0.8	15.5	30	6
c	mr johnson	71	2768	kmc	65	1.4	16.5	32	5
c	mr harish	65	21559	grh	64	0.9	15	33	6
c	mr subramani	60	21817	kmc	55	1.4	18	29	6
c	mr udhayakumar	62	21810	grh	50	2.1	14.5	34	7
c	mr veeran	65	22086	kmc	54	1.5	16	31	8
c	mr xavier	63	21345	grh	65	1.2	19	34	5

IPSSDIF	PREOPQOL	POSTOPQOL	QOLDIFF	PREOPPFR	POSTOPPFR	PFRDIFF	PREOPPVR
18	4	2	2	8.1	14.7	6.6	95
13	4	1	3	8.4	20	11.6	80
7	4	2	2	0	19.6	19.6	
14	4	2	2	7.8	19.6	11.8	110
7	4	2	2	8.8	19.8	11	80
12	4	1	3	6.4	18.9	12.5	110
15	5	3	2	4.8	11.8	7	130
18	4	2	2	7.6	17.2	9.6	110
14	4	1	3	8.4	19.1	10.7	110
19	4	2	2	8.8	20.4	11.6	105
14	4	2	2	8.2	19	10.8	100
18	4	1	3	0	18.3	18.3	
13	4	1	3	9.4	21	11.6	80
21	5	1	4	4.8	14.6	9.8	130
13	4	1	3	7.3	18	10.7	90
18	4	2	2	9	17.1	8.1	85
16	4	2	2	7.6	17.3	9.7	90
13	4	1	3	4.3	16.3	12	120
17	4	2	2	0	17.2	17.2	
15	4	1	3	6.6	20.4	13.8	80
16	4	2	2	0	16.8	16.8	
15	4	2	2	7.6	21.4	13.8	95
18	4	1	3	0	21.3	21.3	
17	4	2	2	6.3	17.8	11.5	100
17	4	1	3	5.8	17.6	11.8	120
15	4	2	2	7.8	15.6	7.8	90

IPSSDIF	PREOPQOL	POSTOPQOL	QOLDIFF	PREOPPFR	POSTOPPFR	PFRDIFF	PREOPPVR
16	4	2	2	9.1	18.4	9.3	90
12	4	2	2	0	22.4	22.4	
21	4	1	3	0	19	19	
20	4	1	3	0	20.7	20.7	
17	4	1	3	0	17.6	17.6	
12	5	3	2	5.1	14.8	9.7	130
21	4	2	2	0	18.2	18.2	
22	4	1	3	8.4	16.8	8.4	100
14	4	1	3	0	18.6	18.6	
20	5	2	3	5.4	13.2	7.8	120
25	5	1	4	6	21.3	15.3	270
13	5	1	4	5.5	17.9	12.4	380
16	5	3	2	6.8	22.5	15.7	120
21	5	1	4	7	19.6	12.6	130
21	4	1	3	5.2	19.6	14.4	90
16	4	2	2	9.6	20	10.4	75
18	4	1	3	0	18.2	18.2	
17	4	2	2	0	17.9	17.9	
17	4	1	3	7.9	20.2	12.3	90
21	4	1	3	0	19.5	19.5	
16	4	1	3	7.9	23.7	15.8	75
14	4	1	3	0	16.2	16.2	
17	4	1	3	0	18.4	18.4	
16	4	1	3	8.6	21.6	13	80
21	5	1	4	11	19.6	8.6	370
22	4	1	3	7.8	19.8	12	284
22	5	1	4	6	18.9	12.9	

IPSSDIF	PREOPQOL	POSTOPQOL	QOLDIFF	PREOPPFR	POSTOPPFR	PFRDIFF	PREOPPVR
24	5	1	4	5	11.8	6.8	100
27	5	2	3	6	17.2	11.2	250
27	5	1	4	7	19.1	12.1	230
23	4	1	3	0	20.4	20.4	
27	5	1	4	0	16.8	16.8	
23	5	2	3	5	21.4	16.4	190
29	5	1	4	0	21.3	21.3	

POSTOPP	PVRDIFF	OPTIME	PREOPHB	POSTOPHB	PREOPRETEN	POSTOPRETE	HOSPSTAY	IRRRIFLUID	
20	75	65	10	10	0	0	5	12	
15	65	55	11.2	11	0	0	4	10	
20		65	10	10	1	0	5	12	
18	92	70	11.8	11	0	0	4	12	
12	68	45	13	12	0	0	5	11	
28	82	62	12.8	11	0	0	4	12	
50	80	50	12	12	0	0	5	10	
40	70	62	11	110	0	0	4	16	
34	76	55	13	13	0	0	5	18	
25	80	50	11.5	10.5	0	0	5	16	
32	68	70	11.3	11	0	0	5	10	
30		45	13.1	12.4	1	0	5	14	
15	65	62	11	10.6	0	0	5	11	
40	90	50	10.8	10	0	0	4	13	
18	72	62	12	11	0	0	5	12	
18	67	55	11.6	11.5	0	0	4	17	
22	68	56	12	11	0	0	5	19	
28	92	55	14	13	0	0	5	16	
25		60	12.3	12	1	0	5	14	
22	58	70	11.3	11	0	0	7	12	
27		65	11	11	1	0	4	16	
0	95	45	12.3	11	0	0	4	18	
20	-20	55	12	11	1	0	5	11	
25	75	50	14	13	0	0	4	12	
25	95	54	10.9	10	0	0	5	12	
24	66	65	12	12	0	0	4	17	

POSTOPP	PVRDIFF	OPTIME	PREOPHB	POSTOPHB	PREOPRETEN	POSTOPRETE	HOSPSTAY	IRRRIFLUID	
22	68	56	13	12.5	0	0	5	12	
		55	12.4	13	1	0	4	11	
18	-18	60	11.5	11	1	0	5	16	
25	-25	70	12.5	12	1	0	4	12	
25	-25	50	12.3	12	1	0	5	10	
35	95	62	11.5	11	0	0	5	15	
45	-45	55	10	10	1	0	5	12	
18	82	70	10.4	10	0	0	5	17	
20		45	12	12	1	0	5	12	
40	80	62	11.4	11	0	0	5	16	
18	252	50	10.4	10	0	0	5	14	
45	335	62	9.8	10	0	0	7	14	
30	90	55	12.1	11	0	0	5	16	
40	90	50	11.4	11	0	0	5	17	
27	63	50	12.3	12	0	0	5	12	
10	65	62	12.4	12	0	0	4	18	
20		55	11	11	1	0	5	15	
30		50	13	12	1	0	4	19	
20	70	65	12	11	0	0	5	17	
27		45	14	13	1	0	4	11	
20	55	55	12.4	12.4	0	0	5	13	
16		50	11	10	1	0	5	16	
30		54	10.8	10	1	0	5	12	
18	62	65	10.3	9.8	0	0	5	11	
160	210	65	12.3	12	0	0	5	15	
32	252	45	12	11	0	0	5	14	
45		55	11	10	1	0	5	18	

POSTOPP	PVRDIFF	OPTIME	PREOPHB	POSTOPHB	PREOPRETEN	POSTOPRETE	HOSPSTAY	IRRRIFLUID	
10	90	50	10.6	10	0	0	5	12	
21	229	54	10.4	10	1	0	5	14	
18	212	65	11.2	11	0	0	5	18	
24		66	12	12	1	0	5	16	
21		56	12.4	12	1	0	5	14	
18	172	64	11	11	0	0	5	15	
20		48	10.4	10	1	0	5	17	

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RELATIONSHIP BETWEEN INTRAVESICAL PROSTATIC PROTRUSION (IPP) AND THE POSTOPERATIVE OUTCOMES IN PATIENTS WITH BENIGN PROSTATIC HYPERPLASIA

Dissertation submitted in partial fulfillment of the requirements of

M.Ch *degree examination*

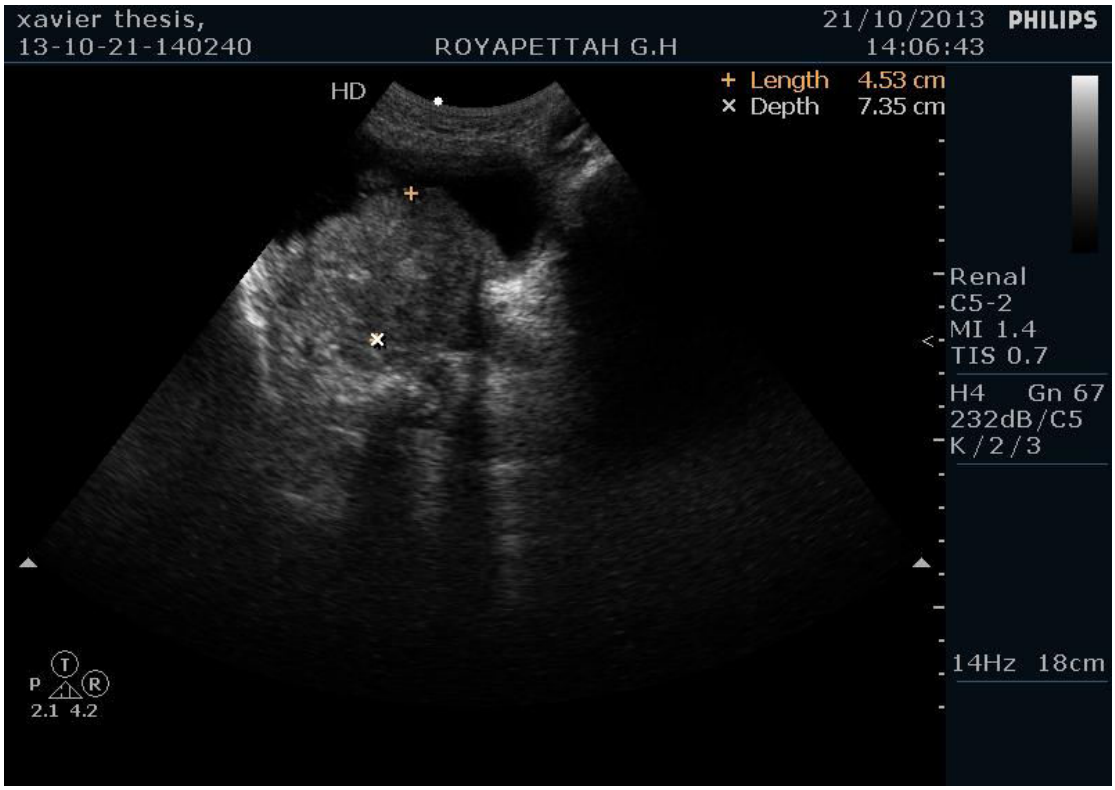
BRANCH IV - UROLOGY

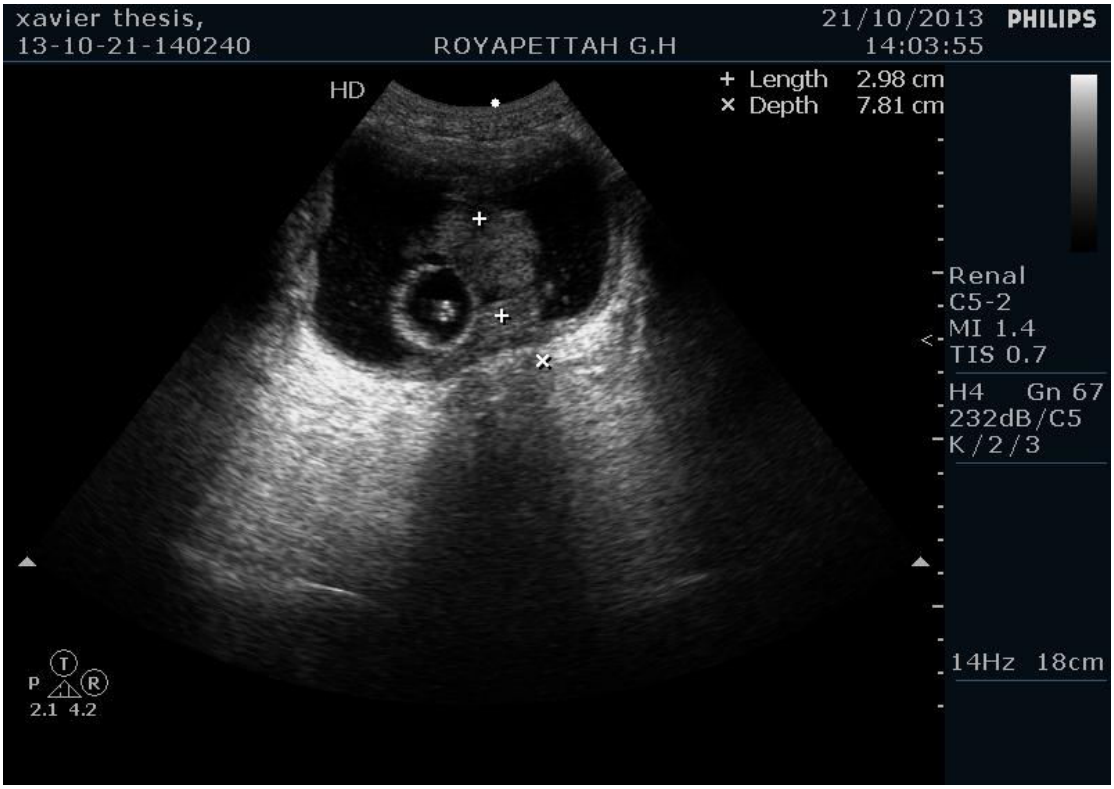
GOVERNMENT KILPAUK MEDICAL COLLEGE

CHENNAI - 600010

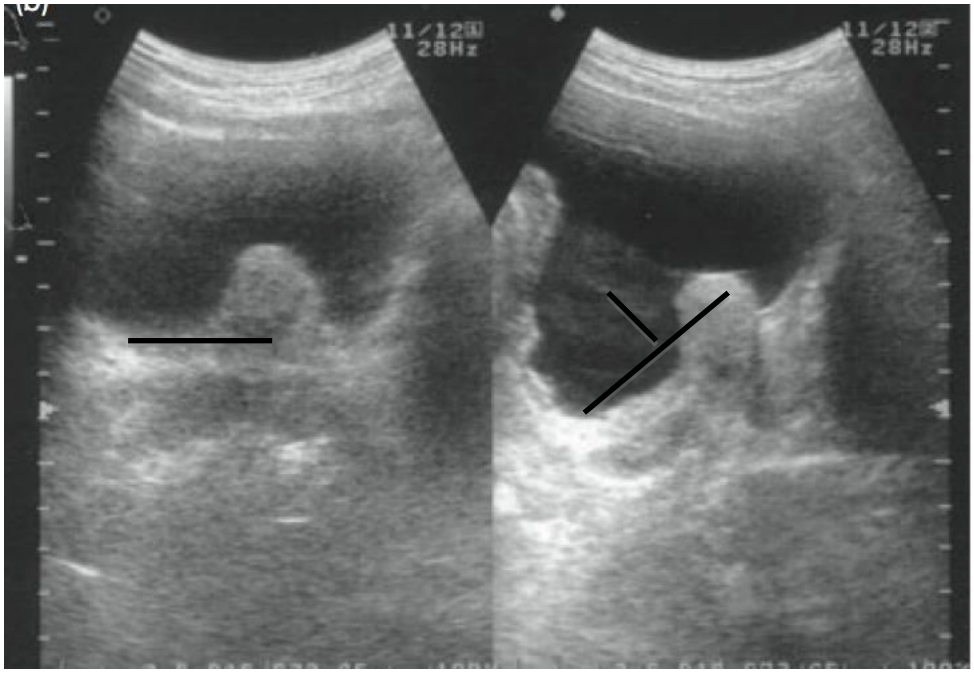
Phillips HD6
7.5MHz Probe

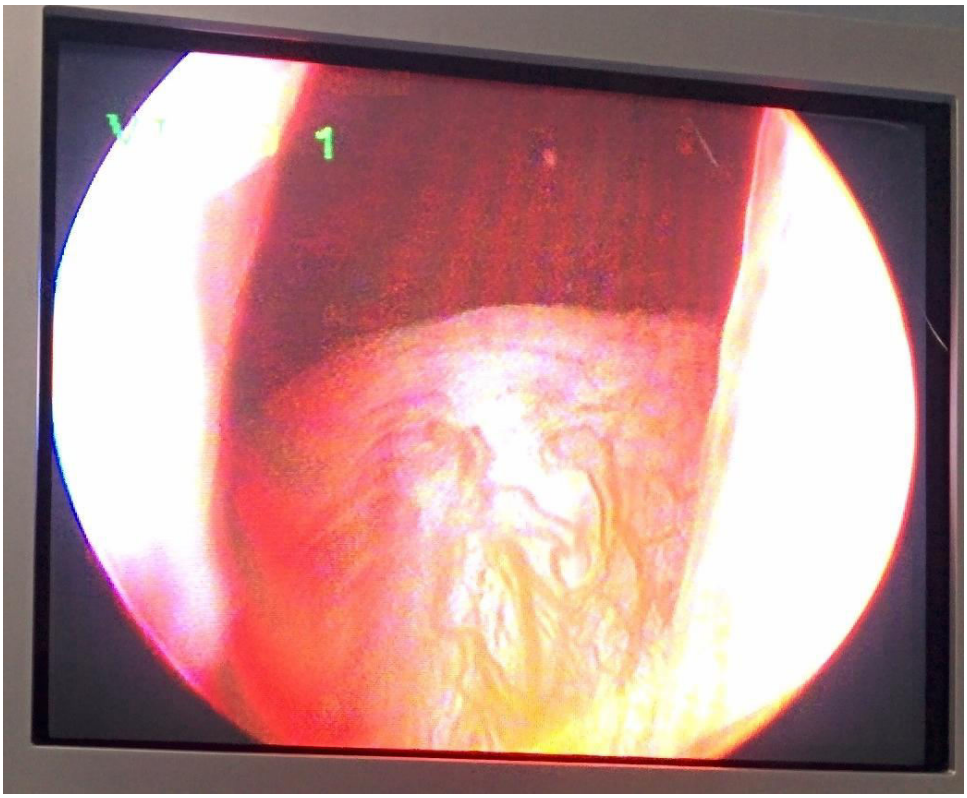
Case 60 Xavier – measurement of IPP





Case 38
Mr.Kannapan





Cystoscopic Image of IVPP